

Figure. Date of onset of cryptosporidiosis cases reported to Health Protection Scotland and date of visit to wildlife center, 2005.

washing facilities were located near the lamb-petting area, and considerable effort was required to locate a handwashing basin in the wildlife center complex. Several alcohol hand sanitizers were located on site, but the microbicidal effects on *Cryptosporidium* spp. are insufficient to prevent infection, especially after direct contact with livestock (4,5).

After publication of the outbreak report, an assessment of hand-washing and hygiene facilities elsewhere in Scotland found them to be suboptimal and that stronger education, regulation, and other control measures were needed to protect the public. Recent *Escherichia coli* O157 outbreaks in England have accentuated the unresolved issues for UK petting farms concerning hand hygiene and zoonotic infections (6).

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Increase in Pneumococcus Macrolide Resistance, USA

To the Editor: Jenkins and Farrell reported an increase in the proportion of macrolide-resistant Streptococcus pneumoniae isolates in the United States (1). They mentioned increased use and inappropriate prescription of macrolides as potential explanations for the increase in macrolide resistance and expressed doubts, stating "which (if any) of these factors might explain the trends here are not clear." Although the spread of antimicrobial drug resistance is a complex issue with many contributing factors, we believe that the role of macrolide use should not be understated.

Several studies in Europe have provided evidence for a relationship between macrolide use and resistance. Macrolide exposure leads to emergence of macrolide resistance on the individual level, and countries in Europe with higher outpatient sales of macrolides have more macrolideresistant pneumococci (2).

Outpatient antimicrobial drug use in the United States has decreased since 1995–1996, especially among children. However, use of azithromycin increased in children, and use of macrolides increased in older patients from 1995–1996 through 2005–2006 (3). In this context, it would be surprising that after this increase, pneumococci would show different characteristics in the United States than in Europe. A 2001 study showed that

increased macrolide use in the United States during 1995–1999 coincided with a doubling of the proportion of macrolide-resistant pneumococci (4), and further increases in macrolide use since 1999 (3) have contributed to the increase in macrolide-resistant pneumococci.

Decreased macrolide use has led to a decrease in macrolide-resistant pneumococci. A yearly seasonal reduction in antimicrobial drug prescribing in Israel was associated with a decrease in the proportion of antimicrobial drug-resistant pneumococci that caused acute otitis media (5). With the introduction of expanded-valent pneumococcal conjugate vaccines, there is promise that drug-resistant pneumococcal disease can be reduced. Nevertheless, judicious use of antimicrobial drugs and a decrease in unnecessary prescriptions, as promoted by the Get Smart: Know When Antibiotics Work (www.cdc.gov/getsmart) campaign, are essential to limiting selection and spread of antimicrobial drug resistance.

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Rapid Antigen Test for Pandemic (H1N1) 2009 Virus

To the Editor: Drexler et al. recently compared the sensitivity of the BinaxNOW Influenza A & B Rapid Test (BinaxNOW; Inverness Medical, Cologne, Germany) with that of a real-time reverse transcription–PCR (RT-PCR) assay specific for influenza A pandemic (H1N1) 2009 virus (*I*). Of 1,838 clinical specimens tested, 221 were confirmed as positive for pandemic (H1N1) 2009 by RT-PCR. When 144 of these 221 specimens were evaluated by using the Binax-NOW, results were positive for only 16 (11%).

At onset of the pandemic, we evaluated the first 135 nasopharyngeal aspirates submitted to the Regional Laboratory of Public Health Haarlem, the Netherlands. We compared the performance of the BinaxNOW for diagnosing influenza A (H1N1) virus by using molecular detection of influenza virus as the reference standard. Samples were analyzed with a general influenza A assay targeting the matrix gene (the RespiFinder assay) (PathoFinder B.V., Maastricht,

the Netherlands [2]) and a pandemic (H1N1) 2009-specific RT-PCR assay targeting the neuraminidase gene (3). We tested 135 patient samples (76 from male patients); mean age of patients was 32 years (range 0-81 years). Samples from 38 (28%) patients had positive results in both RT-PCRs, and samples from 97 (72%) patients had negative results in the matrix gene RT-PCR and neuraminidase RT-PCR assays. Sensitivity and specificity were estimated to be 47% (18/38, 95% confidence interval [CI] 32%-62%) and 95% (92/97, 95% CI 88%-98%), respectively, for the BinaxNOW antigen test. Patients' ages did not significantly differ between rapid test-positive and -negative results.

Our results largely agree with those of Vasoo et al. (4) and the Centers for Disease Control and Prevention (5). Those studies determined that the sensitivity of the BinaxNOW compared with nucleic acid amplification tests is ≈40%. The lower sensitivity observed by Drexler et al. (1) might be because of differences in study type (retrospective evaluation compared with a prospective cohort in our study), sample size, technical factors (with regard to specimen collection, specimen transport, and specimen storage), differences in the test kit, and differences between individual patients (multiple categories of age and stages of illness, differences in virus shedding).

Many clinicians are not aware of the performance of specific test devices and rely on test results to make clinical decisions. Because negative results cannot rule out influenza, this test is of little use in a clinical setting without appreciation of the limitations of the test. However, because the BinaxNOW has reasonable specificity, it might prove useful in clinical or epidemiologic situations in which test sensitivity is not critical, e.g., in facility outbreaks in which multiple specimens are collected to rapidly identify the causative organism.